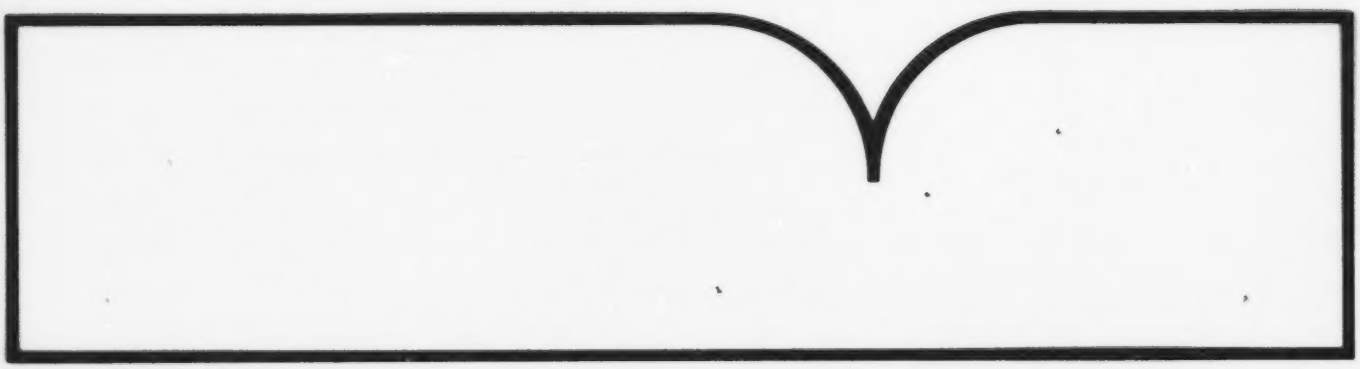
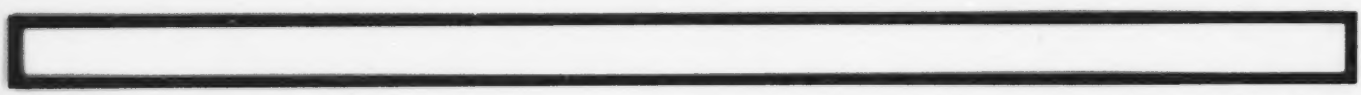
PB88-243506

Review of EPA's (Environmental Protection Agency's) National Dioxin Study

(U.S.) Environmental Protection Agency
Washington, DC

19 Dec 86





U.S. Department of Commerce
National Technical Information Service
NTIS

50272-101

REPORT DOCUMENTATION PAGE	1. REPORT NO. SAB-EC-87-012	2.	3. PB88-243506

4. Title and Subtitle Review of EPA's National Dioxin Study	5. Report Date Dec. 19, 1986 6.
7. Author(s) Executive Committee	8. Performing Organization Rept. No.
9. Performing Organization Name and Address Environmental Protection Agency Science Advisory Board 499 S. Capitol Street, SW Washington, D.C. 20460 (A101F)	10. Project/Task/Work Unit No. 11. Contract(C) or Grant(G) No. (C) (G)
12. Sponsoring Organization Name and Address Environmental Protection Agency 401 M Street, SW Washington, D.C. 20460 (A101)	13. Type of Report & Period Covered Final Report 14.
15. Supplementary Notes	

16. Abstract (Limit: 200 words)

The Subcommittee commended EPA and its personnel for the preparation of a comprehensive, informative and well-written document. With revisions that are identified in the report, the thoroughness of the Study and quality of the data base are scientifically supportable, given the understanding of current knowledge.

The four objectives of the study were: (1) to assess "the associated risks to humans and the environment", (2) a study of the extent of contamination, (3) implementation of site clean-up efforts, and (4) the evaluation of a variety of disposal and regulatory alternatives.

17. Document Analysis a. Descriptors

Executive Committee

b. Identifiers/Open-Ended Terms

c. COSATI Field/Group

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

18. Availability Statement	19. Security Class (This Report)	21. No. of Pages 16
	20. Security Class (This Page)	22. Price A03 · 12.95

SAB-EC-87-012

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

December 19, 1986

OFFICE OF THE ADMINISTRATOR

Honorable Lee M. Thomas
Administrator
U. S. Environmental Protection
Agency
401 M Street, S. W.
Washington, D. C. 20460

Dear Mr. Thomas:

The Science Advisory Board's (SAB) National Dioxin Study Review Subcommittee has completed its review of EPA's draft National Dioxin Study and is pleased to transmit its principal scientific conclusions and recommendations to you. The Subcommittee met in public session on September 8-9 to review the adequacy of the scientific assumptions, methodologies and conclusions of the Study. It subsequently submitted its draft report to the SAB Executive Committee which approved it on December 18th.

The Subcommittee commends the Environmental Protection Agency and its personnel for the preparation of a comprehensive, informative and well written document. Many of the sample collection techniques and the required analytical methodologies were, and still are, state-of-the-art. With some revisions that are identified in this report, the thoroughness of the Study and quality of the data base are scientifically supportable, given our understanding of current knowledge.

The Subcommittee consensus is that the statistical interpretations and extrapolations are, with some corrections noted in the attached report, generally adequate.

There are basically four objectives of the Study. One of the main objectives was to assess "the associated risks to humans and the environment". The other objectives included a study of the extent of contamination, implementation of site clean-up efforts, and the evaluation of a variety of disposal and regulatory alternatives. Considering the logistical and financial constraints, the Study generally met the latter objectives but failed to properly address the risk assessment aspect. To perform risk assessment for one tier and not the others, is inconsistent. EPA should delete the Tier 4 risk assessment altogether to ensure consistency with the entire study. Any specific risk assessment developed for a combustion source or category of sources for any particular EPA decision making activity should undergo peer review.

Reproduced from
best available copy.

The objective of studying the risks of dioxin contamination to the non-human environment was not adequately addressed in the report. To the extent available, results obtained by the EPA (or from other scientifically valid studies) on the bioaccumulation by fish of 2,3,7,8-containing tetra-, penta-, hexa-, and heptachlorodibenzo-p-dioxins relative to that of other dioxin congeners (that do not contain the 2,3,7,8-chlorination pattern) should be included. Results on the fish bioaccumulation of dioxins that are present in sediments and fly ash should also be added if such data are available. Lastly, to the extent that the EPA has such results, information on the toxicity of dioxins to different fish species should be included. At the very least, the inclusion of such a discussion of these ecotoxicological endpoints would demonstrate that the Agency is cognizant of the need to fully study risks of dioxin exposure to the environment.

EPA and the Food and Drug Administration (FDA) propose different values for acceptable TCDD concentrations in fish that are consumed by humans. Since this issue is crucial to the Great Lakes studies, it is difficult to understand why the two agencies have "agreed to disagree". The Subcommittee recommends further discussions with the FDA prior to submitting this study to Congress.

The Study does not clearly state how one characterizes a site with respect to TCDD contamination (i.e. from a sampling perspective). It would be useful to include a sentence or two that would address (a) how many samples are required, (b) whether surface wipe samples or cores are better, and the problems with each approach. With respect to the second sampling method, a straightforward calculation could be made to account for all TCDD being in the upper end, for example, the top 0.5 cm. of a 4 inch core. This calculation would reduce some of the uncertainty as to what constitutes a contaminated sample and thus, by inference, a contaminated site. This comment is directed across several tier efforts where soil or sediment samples were taken.

In general, EPA's limited conclusions appear to be supported by the results of the survey. The Subcommittee believes that additional conclusions may be drawn. While it appears that "off-site" migration of polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) is minimal, there is evidence of widespread accumulation of these compounds at low levels in human tissues.

Given current knowledge, the Agency may have identified most of the significant sources of PCDD and PCDF contamination and/or exposure. Some sources have been treated in more detail than others, but most have been studied to some extent. However, the Subcommittee believes that new sources will probably be discovered in the near future as more knowledge is gained on the various mechanisms of the formation of chlorinated dioxins and furans. This may result in the recognition of previously undetected routes and rates of exposure to humans and the environment.

Based upon the limited survey of selected combustion sources in Tier 4, it is not possible to accurately quantitate the potential environmental input of dioxins and furans from this category for the purposes of the National Dioxin Study. However, for the types of facilities tested, annual loadings can be roughly estimated. The specific sources sampled represent a selection of combustion facilities. While it is not known how many similar facilities exist in the U. S., approximate estimates would provide a range for evaluation.

Because of the large number of combustion facilities, and the increasing reliance on incineration for waste management, Tier 4 sources remain an area of concern. The ubiquitous presence of low levels of 2,3,7,8-TCDD in Americans suggests that combustion sources are responsible at least in part for this general "background" contamination. Investigators in other countries have reached the same conclusion. The Science Advisory Board is currently evaluating this and other issues as they pertain to municipal waste combustion.

The Subcommittee expresses its appreciation for the opportunity to review the National Dioxin Study. Attached to this letter are more specialized technical comments that pertain to certain sections of the Study. The Subcommittee requests a formal Agency response to the scientific advice it has provided, or discussion of the reasons for those issues where the advice is not accepted. It would particularly appreciate this response at the time the Agency formally transmits the final study to the Congress.

Sincerely,

Robert Huggett

Robert Huggett, Chairman
National Dioxin Study Review Subcommittee
Science Advisory Board

Norton Nelson, Chairman
Executive Committee
Science Advisory Board

Enclosure

I. General Scientific Comments on the National Dioxin Study

For each of the seven tiers (representing different sources and routes of potential contamination and exposure) EPA developed a sampling plan that involved two basic stages: (1) selection of sites, and (2) selection of material at the selected sites. The principal statistical issues concern these latter two issues, and the analysis and interpretation of the resulting measurements.

In the first stage, selection varied from complete coverage (Tiers 1, 1a, 2, 2a) through random selection from a list (Tier 7), judgment based selection of Tiers 4 and 5, and combinations of random and judgment selection (Tiers 3 and 6). The within site sampling plans involved primarily judgment, sometimes supplemented with random samples. The sampling plans used in the various tiers appear to represent well chosen compromises between what was convenient (but of limited scientific value because of uncertainty about how the sample compares to the whole) and what was ideal (but not always feasible because of the time and resources required). The findings have, by and large, been allowed to speak for themselves in the report via detailed site-by-site descriptions, with a minimum of formal statistical analysis. The Subcommittee believes this is a scientifically appropriate approach.

Because of the great differences between the various tiers, in terms of both prior knowledge and the sampling plans used in the National Dioxin Study, the degree of uncertainty that remains varies somewhat from tier to tier.

The main text of the report omits much detail and background information, which is acceptable because it enhances the readability of the document. References to the detailed material, however, would be helpful. For example, the discussion of the important question of "What happened to dioxin in the environment?" is found in Appendix B of NDS 3567. A reference to this material in Section 1.3 of the main text would improve the document.

A number of homologues of the PCDDs and PCDFs are present in parts per trillion (ng/kg) concentrations in the adipose tissue of the general population of the United States and other countries. Octachlorodibenzodioxin and octachlorodibenzofuran may reach conentrations in the low parts per billion (ug/kg) range. It appears that those homologues of the PCDDs and PCDFs where the 2,3,7,8 positions are occupied by chlorines are preferentially stored in humans (Graham et al., 1985; Lee and Hobson, 1985; Nygren et al., 1985; Patterson et al., in press; Rappe et al., 1984; Ryan et al., 1985; Ryan and Schecter, 1985; Schecter et al., 1985).

In a series of 59 control adipose tissue samples from the general population, 2,3,7,8 tetrachlorodibenzodioxin could be identified in all samples. The mean concentrations were 6.4 parts per trillion (ug/kg) and the range 1.4-20.2 parts per trillion, ug/kg (Patterson et al., in press). Other investigators have reported similar means and ranges. Thus, like fish, many human adipose tissue samples seem to contain trace amounts of these chemicals (Ryan et al., 1984). These data indicate that widespread low-level contamination has occurred.

Reproduced from best available copy.

Combustion sources produce solids, either as bottom ash or fly ash (much of the latter is collected in the stack by control devices such as bag houses or electrostatic precipitators). Ash may also contain concentrations of dioxins and furans. Therefore, environmental inputs of dioxins and furans from ash represent another potentially significant source of these substances from combustion facilities.

II. Specific Editorial/Technical Comments on the National Dioxin Study

Tier I

I-2,1.1. The first paragraph states that over 500 treated cases of toxic effects were alleged to be associated with the Seveso accident. This statement should be deleted and replaced by one that places into perspective not only data on acute dermititis which had resulted from burns received by the simultaneous release of caustic materials, but also the significance and quality of data on immunotoxicity, neurotoxicity and reproductive effects.

I-8,1.2.2. The Study states that the EPA Carcinogen Assessment Group (CAG) has determined that 2,3,7,8 TCDD is a probable human carcinogen. The Study should contain an explanation of the criteria used by the CAG to determine whether a chemical is a probable human carcinogen and whether these criteria are still the same as they were when the document was written, and whether any more recent evidence further supports or challenges this conclusion.

I-8,1.3.2a. EPA should expand the section on nonhuman toxicity. It fails to mention that some CDD and CDF congeners differ in their ability to produce certain endpoints of toxicity. There are presently insufficient data to determine whether the toxic effects of various congeners are additive, synergistic or antagonistic. Therefore, the sentence, "In addition, more limited data suggests that effects are additive and not synergistic" should be deleted or substantiated with references.

I-8,1.3.2b. The Study should state that incidence of mild chloracne has been observed in humans in Nitro, West Virginia for at least a decade after exposure to 2,3,7,8-TCDD.

I-1T,1.3.3. The Study assumes that only when knowing the concentration of homologous groups is it appropriate to assume equal probability of the occurrence of each of the congeners in the group. This assumption is not supported by data on pyrolysis products.

I-15,1.42. Clarification of the stated detection limits is required. Due to the uncertainity associated with the term, the analytical method quantitation limit may be appropriate in this report. When defining the quantitation limit, the operational constraints (i.e., ten times the signal to noise ratio) should be stated.

The Study should present more detail of the quality assurance/quality control program throughout the test. The reader should be able to readily determine the level of accuracy and precision.

Tier 2

II-9,2.2.3. Was sampling conducted in the spring of 1986?

II-21,2.3. If "little" means less than 1 part per billion (ppb), it should be stated.

II-22, para.2., line 2 and figure 2.3. Under "No further action", the number of sites in the text and the figure do not agree (22 sites text versus 23 sites figure).

II-29 and 30, Table 2.2. Millmaster Onyx and Baird and McGuide. The kinds of samples analyzed are omitted.

Table 2-3. The Centers for Disease Control (CDC) has two types of responses to health questions in contaminated areas: health advisories and health assessments. Not all items listed in table 2-3 are health advisories. The heading of the tables should be changed to "Dioxin sites reviewed by ATSDR/CDC." "Advisory" should be changed to "Recommendation."

Tier III

III-3,3.1.1. The Study should more specifically state the criteria used to determine if a site was contaminated. If one soil sample had less than 1 ppb, or one fish sample had less than 1 ppt of 2,3,7,8-TCDD, was the entire site considered uncontaminated?

III-3,3.1.2. In the discussion of Tier 3 no mention is made of the "...additional 325 potential Tier 3 facilities..." described on page 10 of the final draft report "The National Dioxin Study Tiers 3,5,6,7" (NDS 3567). The existence of these 325 additional potential Tier 3 facilites means that Tier 3 might be twice as large as is indicated in NDS-RTC. The present state of knowledge about these 325 facilities should be clarified to enable the reader of NDS-RTC to have an accurate appreciation of the uncertainty concerning the potential contribution of sources in Tier 3.

III-4, 3.1.3. The numbers given on page III-4 are not consistent. In the first paragraph of section 3.1.3 results, the Study states that "Soil contamination...was found at four of the statistically selected sites." However, in the third paragraph, the Study speaks of "the five statistically selected contaminated sites." (NDC-3567, page 13, suggests that 5 of 41 sites were contaminated.) Similarly, paragraph 2 says that 5 of the regionally selected sites were contaminated, yet paragraph 3 speaks of "...four of the six contaminated regionally selected sites...".

III-5,3.1.3. The statement that "it is estimated that 3 $\pm$ 6 percent of facilities in the FATES data base may be contaminated," needs

clarification. The percentages apparently refer to the population of 312 sites from which the statistical sample was drawn. Thus, they translate to 25 ± 19, or between 6 and 44 contaminated sites. The previous comment (III-3,3.1.2) becomes important here; if similar percentages apply to the 325 "additional potential Tier 3 facilities," then the number of contaminated sites is roughly twice as large. If there is reason for confidence that similar percentages do not apply because of specific relevant differences between the 312 sites and the 325 "potential" sites, this should be made clear. Otherwise, the possibility that the "8 ± 6 percent" applies to more than 600 sites (not only to the 312) should be acknowledged.

The assumption that the seven missing eligible sample sites are not contaminated should be justified somewhere. The parenthetic explanation, "(based on their physical characteristics)", is insufficient. Can EPA provide a reference to where a fuller analysis and explanation justifying the assumption can be found? If not, the cautious reader may well decide that the higher figures given in Table C-2 of NDS-3567 (p. C-4), i.e. 10 ± 8 percent (31 ± 25 contaminated sites), are more reasonable; and the skeptic might even choose the other extreme assumption of all seven missing sites contaminated, leading to 20 ± 10 percent, or 31 to 93 sites contaminated. It might be best to estimate the total number of sites like the seven missing ones (where there has been extensive grading and/or paving) and frankly acknowledge that only educated guesses can be made about the amount of contamination at such sites. The population of 312 would be reduced by this amount, and new estimates of the percent contaminated would be required.

One additional point is that the estimation is not the number of sites that are contaminated, but the number that would be found to be contaminated if they were all sampled using the procedures and techniques of this Study. For every site at which contamination is present there is some probability that EPA's investigation procedures would lead to a false negative finding because of variability involved in choosing where to take samples as well as because of processing and analytical errors. We expect that this probability is low for heavily contaminated sites, but not necessarily low for marginal ones. On the other hand, the probability of false positives would appear to be much lower and might well be negligible.

III-18,3.1.5 The second conclusion does not seem to agree with page II-7, where it is stated that 13 sites from Tiers 3-7 have been referred. The statement given in section 3.1.5 is not at all clear; does it imply that all remaining Tier 3 facilities have been referred?

Tier 4

In order to assess the risk of combustion related airborne TCDD to humans, one needs to know how much TCDD is emitted into the atmosphere at a representative number of sources. Experience from sampling trace metal emissions from sources such as coal fired steam plants, incinerators.

and smelters has taught the scientific community that, while annual loadings can be roughly estimated for the types of facilities tested, each source exhibits a wide range of temporal variability and each type of source can be very much different in the chemical characteristics of its emissions. Some of the shorter term variability may be "smoothed" by using a longer period of sampling. However, the Subcommittee believes that the 13 combustion sources do not adequately represent the rest of available TCDD air emission sources in the U. S. The numbers do not, for example, permit a calculation of the annual U. S. TCDD emissions into the atmosphere from stationary combustion sources. A second step in calculating human health risks involved modeling the atmospheric dispersion of the emitted TCDD using some unspecified model. This kind of exercise has been quite common for SO_2 and SO_4. What these modeling efforts have taught is that, with appropriate meteorological data, it is possible to do a reasonably good job of estimating the yearly average air concentration, but daily, weekly, or even monthly predictions (especially "plume touchdowns") are not satisfactory. Hence, it would be very doubtful that the "average" calculated TCDD air concentration accurately reflect dosage to humans.

Combustion sources produce solids, either as bottom ash or fly ash (much of the latter is collected in the stack by control devices such as bag houses or electrostatic precipitators). Ash may also contain concentrations of dioxins and furans. Therefore, environmental inputs of dioxins and furans from ash represent another potentially significant source of these substances from combustion facilities.

Because of the large number of combustion facilities, and increasing reliance on incineration for waste management, Tier 4 sources remain of concern. The ubiquitous presence of low levels of 2,3,7,8-TCDD in Americans suggests that combustion sources are responsible at least in part for this general "background" contamination. This has been the conclusion of investigators in other countries as well.

It is not clear whether the risk calculations from stack emissions are based on CDDs and CDFs in the gas phase only, or whether they also include particulates. If they are based on particulates, the human dose would depend on the particle size, and only a small portion of these materials would actually be inhaled.

Sampling results and physico-chemical data indicate that TCDD in air should be mainly associated with micron-to-submicron aerosols. Yet the air dispersion of combustion related TCDDs was modeled as a vapor. Atmospheric removal efficiencies of vapors and particles may differ considerably, rendering results from the model calculations in the document suspect.

While it is reasonable to expect that most of the emitted TCDD is associated with 1-2 micron or submicron sized particles, it is not clear at all how available this compound is to transfer into the human lung.

These weaknesses in arriving at dosage estimates increase the scientific difficulty of performing risk assessment. To develop a risk assessment for

this tier and not the other tiers creates an inconsistency for the entire Study. EPA should, therefore, delete the Tier 4 risk assessment entirely. Any specific risk assessment developed for a combustion source or category of sources for any particular EPA decision making activity should undergo peer review.

Tier 5

III-19,3.2.2. A major source of uncertainty in Tier 5 concerns the size of the tier. However, this becomes a matter of practical importance only if the levels of contamination in Tier 5 are great enough to be of concern.

The soil samples were comprised of a four inch plug of material. If the compounds of interest were in only the first centimeter or less, EPA could have greatly underestimated the reported concentrations in the fraction most biologically available to humans (hand-to-mouth, reintrainment of dust). EPA data which shows dioxins to be relatively immobile in soils suggest that this may be the case. In the absence of more specific information, the Agency should develop calculatons that assume only 1 to 10 millimeters contamination, and it should examine the findings and conclusions relative to the results.

III-28,3.2.4. The fifth finding may be misleading because the text implies only 4 contaminated sites. Also III-20, 3.2.3 states that 15 sites were contaminated, not 13.

III-35, Table 3.2. Some A. R. Desha Data are missing. To the table should be added: Soil, 465 samples, 1 positive, 3 ppt 2,3,7,8-TCDD.

Tier 6

III-30, 3.3.2. EPA did not consider one chemical product, the production of chlorophenol from chlorobenzene, although there is a chemical site in Niagara Falls, New York where a significant degree of contamination with dioxins, including 2,3,7,8-TCDD, is asociated with this product.

III-[redacted], 3.3.4. Findings and conclusions should be examined relative to the Subcommittee's comments presented above.

Tier 7

"Background sites" in the Tier 7 effort is probably not a good designation. The word "background" has a different implication than what the authors tried to accomplish with their site selection strategy.

The TCDD data on fish is difficult to interpret in terms of what extrapolations can be made to other fish. Data are now available on other chlorinated hydrocarbons in fish, especially with respect to different age, weight, sex, time of year, and species. Concentration distribution functions could be constructed from these data and used in conjunction with the TCDD data for a more meaningful interpretation.

A perusal of the physico-chemical data in the main document as well as in the tier background documents indicates that some of these values could be updated. Recent measurements on the aqueous TCDD solubility and vapor pressure at 25°C are: C_s = 6 x 10^{-11} moles^s/L = 1.93 x 10^{-5} mg/L = 19.3 ng/L (ppt); p^s = 7.4 x 10^{-10} torr = 9.47 x 10^{-13} atm. This leads to a Henry's Law constant, H = 1.6 x 10^{-5} atm-m^3moles^{-1}, which is 8-10 times different than the one reported in these documents.

III-43, 3.4.2. Fish. More detailed information on the sampling protocol should be provided to clarify that 4 fish samples per site were analyzed and, if the first one (a whole, bottom feeding fish) was negative, the other 3 samples were not analyzed. (See the National Dioxin Study, Tiers 3,5,6, and 7, April, 1986, Page 41, para. 3.) Additionally, EPA should specify the criteria for a positive sample for TCDD contamination (greater than 1 ppt?).

III-45, 3.4.3. para. 2. The Study should state whether the maximum concentrations presented were for whole fish or filets.

III-45, para. 3. Was it demonstrated statistically that fish sampled from the Great Lakes were larger than those sampled from inland waters?

III-46, para. 2. The statement on page III-46 appears to take exception to the acceptable level of 25 ppt TCDD in fish (under certain restrictions) set by the FDA. This page indicates that consumption of fish containing 25 ppt TCDD may pose an upper bound human cancer risk of 25 X 10^{-4}, a high risk according to current regulatory practice. If the Agency maintains its position that consuming fish containing 1 ppt of fish may pose an unacceptable risk (1 x 10^{-5}) the implications to freshwater commercial and sport fishery may be far reaching.

II-48, 3.4.5. The Study acknowledges that the fish data may be a cause for human health concern, but it ignores the potential significance of these data in estimating the prevalence of 2,3,7,8-TCDD in the environment. The fish tissue data may actually represent a better indicator of the prevalence of 2,3,7,8-TCDD than the soil samples, particularly since the top five inches of soil were collected and blended. The various bodies of water from which fish were sampled in this Study may serve as integrators of the inputs into their respective watersheds and the fish, in turn, serve to integrate the bioavailable 2,3,7,8-TCDD which has accumulated over time in water and sediments. The final report to Congress should discuss the relevance of the fish data in estimating the distribution of 2,3,7,8-TCDD and the apparent inconsistencies between these data and the soil data.

III-53, Figure 3.6. EPA should change the title of this figure to "Distribution of 2,3,7,8-TCDD Concentrations in Whole Fish Samples From Different Locations".

III-59 to III-71, Tables 3-6 and 3-7. Results for Great Lakes fish are not included in any of the tables, whereas fish from statistically selected and regionally selected sites are included. This omission should be corrected. Also, if possible, the type of fish species sampled at each site should be added to the tables.

Toxic Equivalency Factors

The Science Advisory Board formed a Dioxin Toxic Equivalency Factor Subcommittee to conduct a review of the assumptions and principles used by EPA in developing interim toxic equivalency factors for mixtures of CDDs and CDFs. Drs. Huggett, Kimbrough, Neal and Silbergeld participated in that review which occurred on September 8-9, 1986. The National Dioxin Study Subcommittee did not, therefore, conduct a separate scientific review of EPA's toxic equivalency factor methodology.

The Subcommittee recognizes that agencies need to regulate human exposures to mixtures of chlorinated dioxins and furans, some components of which have not been examined for chronic toxicity. The Subcommittee recommends that the Agency use toxicity equivalence factors as an interim risk management tool, clearly stating in the document that the procedure contains a number of limitations.

Research

VI-6,6.4. The Study states that studies are being proposed to enable predictions of dioxin uptake into plants and thereafter into the food chain. Most scientists recognize that plants do not take up these types of compounds, with the possible exception of root vegetables.

VI-8, 6.5. EPA should identify a number of other studies that were funded using Superfund resources. These include:

1. Missouri Dioxin Study

Chemical wastes, including dioxin orginating from the NEPACCO/ Syntex plant in Verona, Missouri, contaminated some 36 sites in Missouri. Present and former residents at one site (the Quail Run Mobile Home Park, as well as a group of unexposed persons for comparison) were given a comprehensive examination to provide information on possible health effects from environmental exposure to dioxin. Results have been published in the Journal of the American Medical Association (JAMA) in 1986.

2. In a subgroup of this study, a medical follow-up examination was completed for study participants who were found to have evidence of immunologic abnormalities and who elected to participate in the follow-up examinations. Analysis of the test results is in progress.

3. Missouri Dioxin Adipose Tissue Study

The purpose of this study is to measure adipose and serum levels of dioxin (TCDD) in populations potentially exposed to dioxin. Study authors think that body burden measurements of TCDD will provide important information concerning the toxicology and epidemiology of chronic environmental exposure to TCDD. The study will examine TCDD levels in adipose tissue and serum from individuals exposed to dioxin. Preliminary results were presented at Dioxin '86 in Fukuoka, Japan and have been published in Patterson et al., JAMA, 256: 2683-2686, 1986.

Reproduced from best available copy.

4. Reproductive Outcome Study at Missouri Dioxin Sites

This study is designed to provide information concerning possible adverse reproductive outcomes related to long-term environmental exposure in 9 residential areas in Missouri where waste oil mixed with dioxin was sprayed on roads for dust control beginning in May 1971. In phase I, the rates of adverse reproductive outcomes for the approximately 400 births from 1971 to 1982 to women exposed to dioxin will be compared to rates for an age and race-matched control group. In Phase II, the medical records of all obstetrical and pediatric hospitals in the state will be surveyed to determine the background rate of malformations for Missouri.

As of June 25, 1986 the authors have completed all data collection for phases I and II. Analysis of the data collected during Phases I and II, in terms of exposure to TCDD, has begun. The authors have prepared a first draft of the Phase I report and submitted it to Centers for Disease Control staff for review. Preliminary analysis of the Phase I data has not demonstrated statistically significant rates of abnormalities among births in women exposed to dioxin. Plans for a Phase III Quality Control sample have been completed; medical records for 50 infants in 15 hospitals will be selected at random for a complete medical records review as quality control for Phase I and II data.

5. NIOSH/CDC Mortality Study of Workers Exposed to Dioxin

The purpose of this study is to determine the mortality outcome of U. S. production workers exposed to dioxin contaminated chemicals. This includes approximately 6,000 workers from 14 facilities throughout the U. S. Investigators are testing 4 specific null hypotheses that there is no association of exposure to dioxin contaminated products and death due to soft tissue sarcoma, lymphoma, stomach cancer and liver cancer.

Through January-March, 1986, a submission was made for vital status follow-up to the National Death Index (NDI) and to the Health Care Financing Administration (HFCA). The review of medical records for information on chloracne has been completed for all 14 plants. Investigators have also received large amounts of data concerning analyses of dioxin from one to two sources that were slow in responding. One major source of analytic data has not yet produced the information, though it is expected to be received during the third quarter of 1986.

6. Pilot Study

The CDC's Center for Environmental Health has conducted a pilot health study in residents who predominantly came from Times Beach, Missouri. (Arch. Environ. Health 41:16-22, 1986).

References

Graham, M., Hileman, F., Kirk, D., Wendling, J. and Wilson, J. (1985) Background human exposure to 2,3,7,8-TCDD. Chemosphere 14:925-928.

Lee, L. E., and Hobson, L. B. (1984). 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) in body fat of Vietnam veterans and other men. In: Chlorinated dioxins and dibenzofurans in the total Environment II (L. H. Keith, C. Rappe and G. Choudharary Eds.), pp. 205-214, Boston.

Mygren, M., Hansson, M., Rappe, C., Dommellof, L., and Hardell, L. (1985). Analysis of polychlorinated dibenzo-p-dioxins and dibenzofurans in adipose tissue from soft-tissue sarcoma patients and controls. Preprint Extended Abstract presented at the American Chemical Society Meeting, Miami 160-163.

Patterson, D. G. Jr., Hoffman, R. E., Needham, L. L., Roberts, D. W., Bagby, J. R., Pirkle, J. L., Falk, H., Sampson, E. J., and Houk, V. N. Levels of 2,3,7,8-tetrachlorodibenzo-p-dioxin in adipose tissue of exposed and control persons in Missouri-An interim report (JAMA in press).

Rappe, C., Nygren, M., Lindstrom, G., and Hansson, H. (1985). Dioxins and dibenzofurans in human tissues and milk of European origin. Abstract 76 published in the Proceedings of the 5th International Symposium on Chlorinated Dioxins and Related Compounds, Bayreuth, Germany.

Rappe, C., Bergovist, P. A., Hansson, M., Kjeller, L. O., Lindstrom, G., Marklund, S., and Nygren, M. (1984). Chemistry and analysis of polychlorinated dioxins and dibenzofurans in biological samples. In Banbury Report 18. Biological Mechanisms of Dioxin Action (Poland, A., and Kimbrough, R. D., Eds.), pp. 17-24, Cold Spring Harbor Laboratory.

Rayan, J. J., Lau, P. Y., Pilon, J. C., and Lewis, D. (1984). 2,3,7,8-Tetrachlorodibenzo-p-dioxin and 2,3,7,8-tetrachlorodibenzofuran residues in Great Lakes commercial and sport fish. In: Chlorinated Dioxins and Dibenzofurans in the Total Environment (Choudhary, G., Keith, L. H., and Rappe, C., Eds), pp. 87-97, Boston.

Ryan, J. J., Lizotte, R., and Lau, B. P. Y. (1985). Chlorinated dibenzo-p-dioxins and chlorinated dibenzofurans in Canadian human adipose tissue. Chemosphere 14, 697-706.

Ryan, J. J. and Schecter, A. (1985). Distribution of dioxins and furans in human autopsy tissues from the general population. Preprint Extended Abstract, presented at the American Chemical Society Meeting, Miami, pp. 158-159.

Schecter, A., Ryan, J. J., Lizotte, R., Sun, W. F., Miller, L., Gitlitz, G., and Bogdasarian, M. (1985). Chlorinated dibenzodioxins and dibenzofurans in human adipose tissue from exposed and control New York State patients. Chemosphere 14, 933-937.

U. S. ENVIRONMENTAL PROTECTION AGENCY
SCIENCE ADVISORY BOARD
NATIONAL DIOXIN STUDY REVIEW SUBCOMMITTEE

Chairman

Dr. Robert Huggett
College of William and Mary
Chairman, Department of
Chemical Oceanography
Virginia Institute of
Marine Science
Gloucester Point, Virginia 23062

Dr. Anders W. Andren
Water Chemistry Laboratory
660 N Park Street
University of Wisconsin
in Madison
Madison, Wisconsin 53706

Dr. Renate Kimbrough
Centers for Disease Control
Center for Environmental Health
1600 Clifton Road
Atlanta, Georgia 30333

Dr. Charles Norwood
4958 Escobedo Drive
Woodland Hills, California 91364

Dr. Richard Peterson
School of Pharmacy
425 N Charter Street
University of Wisconsin
Madison, Wisconsin 53706

Dr. Richard Royall
Professor
Department of Biostatistics
Johns Hopkins University
615 North Wolfe Street
Baltimore, Maryland 21205

Director

Terry F. Yosie, Director
Science Advisory Board
U. S. Environmental Protection Agency
401 M Street, S. W.
Washington, D. C. 20460

Dr. Kenneth Jenkins
Director, Molecular Ecology Institute
California State University
at Long Beach
Long Beach, California 90807

Dr. Robert Neal
President, Chemical Industry
Institute of Toxicology
Post Office Box 12137
Research Triangle Park, NC 27709

Dr. Pat O'Keefe
New York State Department of Health
Division of Environmental Services
Wadsworth Center for Laboratories
and Research
E. S. B.
Albany, New York 12201

Dr. James Petty
Fish and Wildlife Service
Columbia National Fisheries
Research Laboratory
Columbia, MO 65201

Dr. Ellen Silbergeld
Senior Scientist
Environmental Defense Fund
1616 P Street, N. W.
Washington, D. C. 20036